Case report
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A Hepatic Encepholopathy Case By Abusing Synthetic Cannabinoid and Ecstasy

Sentetik Kannabinoid ve Ekstazinin Kötüye Kullanıldığı Bir Hepatik Ensefalopati Olgusu

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ABSTRACT

The increasing prevalence of illicit drugs abuse is a significant social and medical problem. This situation can cause various results, ranging from simple intoxication to fatal organ failure. The most common illicit is ecstasy (MDMA) and marijuana (cannabis). In this case report we tried to present a hepatic encephalopathy case due to using these drugs.

Keywords: *Ecstasy, a synthetic cannabinoid, hepatic encephalopathy*

ÖZET

Önemli bir sosyal ve tıbbi sorun olan, yasadışı uyuşturucu maddelerin istismarın prevalansı artmaktadır. Bu durum basit zehirlenmeden ölümcül organ yetmezliğine kadar çeşitli sonuçlara neden olabilir. En yaygın yasadışı maddeler ekstazi (MDMA) ve esrardır (cannabis). Bu olgu sunumunda bu maddeleri kullanmaktan kaynaklanan hepatik ensefalopati olgusunu sunmaya çalıştık.

Anahtar kelimeler: *Ekstazi, Sentetik kannabinoid, hepatik ensefalopati*

INTRODUCTION

The use of drugs without the doctor's prescription, except for treatment, is named drug abuse or misuse of drugs. These drugs are; caffeine, tobacco, alcohol, morphine and its derivatives, benzodiazepines, barbiturates, amphetamines, hashish, heroin, cocaine, volatile solvents, and bonsai (1). These agents with various toxic effects can lead to many problems, from simple deprivation symptoms to death (1). In this article, we have tried to present a case that developed hepatic encephalopathy based on toxic hepatitis following the use of an intensive amount of ecstasy and bonsai.

CASE

A seventeen-year-old male patient was brought to the emergency with the ambulance due to complaints of yellowing in skin color, nausea, vomiting, meaningless speech, difficulty in walking, and uncontrolled movements.

According to the anamnesis; sudden nausea and vomiting started one day before the application, his skin color changed to dark yellow-green, and on that day, these symptoms were accompanied by meaningless speeches. He had uncontrolled movements with a big amplitude on all extremities, especially in the arms. It was reported that the patient took five MDMA pills and one bonsai tablet, and no other medicine or herbal substances. At his admission to the emergency service, he had a poor and degraded general appearance, and he also had poor self-care with an anxious mood. He was conscious of the disoriented speeches and movements. His maximum heart rate was 110/min, respiratory rate was 26/min, blood pressure was 112/67 mm Hg, and oxygen saturation was 99%. He had dark yellow-green skin. There was horizontal nystagmus. There were uncontrolled choreiform movements with high amplitude in four extremities, especially the upper extremity. Also, there were asterisk-like movements and essential tremors in the hands. The other systemic examination (and also the neurological examination) was normal.

In the serum biochemical examinations; white blood cell was 24.300/mm3, aspartate transferase was 1182 U/l, alanine aminotransferase was 1921 U/l, lactate dehydrogenase was 576 U/l, creatinine kinase was 478 U/l, total bilirubin was 18.8 mg/dl, direct bilirubin was 9.72 mg/dl, C-reactive protein was 9 mg/dl, procalcitonin was 1.82 mg/dl, active partial thromboplastin time was 36 seconds, prothrombin time was 21,6 seconds, and INR was 1.89. In urine toxicological analyses, amphetamine was positive. The other laboratory findings were normal.

With the present findings in consideration, the patient was preliminarily assessed with acute hepatitis, hepatic failure, and hepatic encephalopathy. To make the definitive diagnosis of acute hepatitis etiologically, viral hepatitis, Ebstein Barr Virus, and cytomegalovirus serologies were detected normally. To rule out autoimmune hepatitis, autoimmune antibodies were studied, and it was assessed as being normal. The serum ammonia level, which was checked to rule out metabolic diseases, was normal. Blood ceruloplasmin level was determined as normal for Wilson's disease. The abdominal and Doppler ultrasound imaging showed no abnormalities. The current liver disease of the subject was assessed as toxic hepatic related to the agents he took. As it was likely that his encephalopathy could be due to cerebral edema, a fundus examination was conducted and determined as normal. Magnetic resonance images of the brain were determined to be normal. The electroencephalographic waves showed us an encephalopathic pattern. The findings were evaluated as hepatic encephalopathy. The subject was examined together with the hepatology department, and as a general supplementary treatment, Vitamin K, Ursodeoxycholic acid, and N-Acetylcysteine treatments were started. A diet rich in carbohydrates and poor in protein was initiated for the subject, and he was put under observation. He was transferred to the liver transplantation center with the diagnosis of acute liver failure.

DISCUSSION

From the 1990s onwards, the use of ecstasy, a sort of stimulant and hallucination, has become widespread in Europe and the USA. Likewise, it has started to be used at an increasing rate in our country, as well (2,3). The use of ecstasy is so common in some regions that Andreu et al. reported that ecstasy is the second most common reason in subjects diagnosed with toxic hepatitis under the age of 25 (4). Hepatotoxicity due to ecstasy appears to be independent of dosage and frequency of use. In alignment with the literature, our subject developed a clinical picture of liver failure after the intake of just five pills. The issues with hepatotoxicity due to ecstasy can both recover spontaneously and follow a very poor process, even to death. Liver transplantation was done to some subjects with a fulminant process (4,5). The most extensive series revealing the results of liver transplantation in fulminant hepatitis due to ecstasy belongs to Brauer et al., who compared the subject to whom they applied liver transplantation for fulminant hepatitis due to ecstasy with the other nine similar subjects in literature (5). In our issue, the developing hepatotoxicity progressed fulminant and brought about the need for liver transplantation.

In Turkey, bonsai use is on the increase, like ecstasy (6). However, as pharmacology laboratories study the drug levels usually with the CEDIA method, bonsai is not determined in urine and blood samples though there is a story of bonsai use, which hides the real abuse rates of these agents (7). One of the primary problems with analyzing and recognizing such new-generation drugs is that. There are a lot of kinds of isomers and their derivatives, and their biochemical structures are often changed for them to escape the screening tests (8). This leads to difficulty in the detection of new-generation drugs. Their negative effects are convulsions, anxiety, aggressiveness, muscle rigidity, and confusion (8). The cause of profound agitations, anxiety, epileptic fits, and convulsion observed in these agents is the agonist activity and GABA enzyme inhibition (8). Neurological symptoms in our subject were nystagmus and choreiform movements, which we think are due to GABA inhibition. Some other cases have been reported for acute kidney failure, acute visual loss, Wernicke syndrome, and liver failure due to synthetic cannabinoids (9). According to the UN report of 2010, as the components of synthetic cannabinoids are many and various, the determination of its specific effect is difficult. Therefore, there is no known specific antibody or treatment for it, except for symptomatic treatments (9).

As a result, the increased use of both ecstasy and bonsai will become more and more critical for public health in the near future. Serious problems are caused by the high-distribution volume feature of synthetic cannabinoids. They can cause prolonged and exaggerated effects due to showing accumulation caused by their lipophilic component, the presence of increasing isomers and their derivatives, and the inability to determine the agents in scanning tests. Although the subject who applied to our emergency service showed indications of intoxication, the use of synthetic cannabinoids should first be thought of for the topics that prove to have a negative drug panel. Then symptomatic treatment should be started immediately. Considering that symptoms may appear in the late period, the follow-up/observation periods should be kept as long. To prevent probable deaths, pharmacology laboratories should be updated quickly. In the subjects who apply to the emergency services due to acute liver failure table, the use of these agents should be questioned and considered as an etiologic agent, especially in the adolescent group.

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